and streptococcus vaccines do not seem as effective in most instances as the milk injections.

We have briefly reviewed our experience with non-specific protein therapy.

CONCLUSIONS

1. Non-specific protein therapy is curative in kerionic ringworm of the beard and scalp.

2. Certain resistant syphilitic lesions respond to non-specific protein therapy after the so-called specific drugs have failed to influence them.

3. Nonspecific foreign protein therapy, while not necessarily curative in many chronic dermatoses, has a definite place in the treatment of them.

384 Post Street.

DISCUSSION

ERNEST DWIGHT CHIPMAN, M. D. (350 Post Street, San Francisco)—The paper by Miller and Epstein is deserving of much praise because it is a definite contribution that is calculated to stimulate dermatologic research in the field in which it is most needed.

There are numerous dermatoses the cause of which we have never discovered. Some of these we handle, after a fashion, empirically. There are others in which we may or may not know the cause that defy our best therapeutic efforts.

While there may be details in which some of us may differ, it seems to me that the paper should be considered in its entirety as representing a sincere effort to make progress where progress is required. The merit of the paper is as great in what it suggests as what it states; it even holds out hope against that bugbear of dermatologists, lupus erythematosus. For all its conservatism and modesty, the paper is most stimulating.

❦

GEORGE D. CULVER, M. D. (323 Geary Street, San Francisco)—In discussing "Non-Specific Protein Therapy in Dermatology," as presented by Doctor Miller and Doctor Epstein, I wish to express my appreciation of the careful manner in which they have prepared their subject. So much depends upon therapy in dermatology that whatever proven additional aid can be given to this part of the subject becomes of untold value.

Many of us have noted in our experience that kerionic ringworm, whether of the beard or scalp, that may have the most formidable appearance may be the most amenable to treatment. I recall a number of instances, one of an adult with kerion of the nape of the neck extending into the scalp that made a spectacular recovery. This recovery might well be explained by the fact that with a marked added pyogenic infection there was a rapid absorption of non-specific proteins.

The assistance given one in treating that most obstinate syphilitic manifestation, interstitial keratitis, is a great boon to the syphilologist.

Doctor Miller and Doctor Epstein deserve our commendation.

❦

GEORGE F. KOETTER, M. D. (1136 West Street, Los Angeles)—Non-specific protein therapy is of definite value in the treatment of certain dermatoses.

Psoriasis, lupus erythematosis, and other recalcitrant dermatosis of unknown etiology resist our best therapeutic measures.

I have observed remarkable, apparently curative results in the treatment of dermatitis herpetiformis by the intravenous injection of autoserum or peptone.

Miller and Epstein deserve praise for their concise, stimulating presentation of this subject.

❦

KENDAL FROST, M. D. (523 West Sixth Street, Los Angeles)—Doctor Miller's and Doctor Epstein's resumé of the situation is indeed noteworthy and deserves careful study.

I might add that many cases of psoriasis respond, at least temporarily, to non-specific protein therapy. I have used it chiefly in the form of autogenous blood or autoserum. Many cases which have resisted local applications of all sorts will respond readily after a series of treatments of this sort.

We can well do with more work of the painstaking and modest type of Miller and Epstein.

POLIOMYELITIS INVOLVING THE URINARY TRACT

By HENRY A. R. KREUTZMANN, M. D.
San Francisco

THAT lesions of the spinal cord frequently involve the bladder, and may do so quite early in the course of the disease, in fact sometimes before any pathology is noted in the central nervous system, is becoming more and more evident.

Nitze was the first to call attention to the changes in the bladder which occur in cerebrospinal lues. The cystoscopic picture in this disease is a characteristic one, and the term "tabetic bladder" is now of common usage.

Bladder changes have been observed in many other diseases involving the central nervous system, and in these cases the term "spinal-cord bladder" is used.

BLADDER CHANGES IN SPINAL-CORD DISEASES

The various cord conditions in which bladder changes have been noted and reported are: tabes dorsalis; postapoplectic conditions; gumma of spinal cord; tumors of spinal cord; psammoma of the dura mater; fractures of the spine; injuries of the cord; hematomyelia; syringomyelia; spina bifida; pernicious anemia.

The symptoms in spinal cord bladders form a distinct group which are quite typical in most cases. In the early stages there is an irritation of the nerves and a spasm of the vesical sphincters so that there may be difficulty of urination or even acute retention. As the nerves become paralyzed, a different picture presents itself. In the later stages there is hesitancy in urination and the stream lacks force. At times the patient complains of nocturnal enuresis.

If the bladder is free of infection the patient's only complaint may be great straining on voiding and weakness of the urinary stream. However, if infection is present we have the added symptoms of frequency, urgency, dysuria, and nocturia.

The terminal stage, associated with entire destruction of the spinal cord may produce either paralysis with automatic emptying of the bladder or complete incontinence.

Examination of these patients reveals certain findings common to most of them. The external genitalia are flabby and show marked loss of tone. There is considerable straining on the part of the patient before the urine begins to flow. The stream is of poor force, with dribbling at the end. Catheterization causes little or no discomfort, due to diminished sensation. On passing a soft rubber catheter some obstruction is felt at the bulbomembranous junction as a result of the constant spasm of the external sphincter. When the eye of the instrument enters the posterior

urethra urine begins to flow. The bladder contains a varying amount of residual. While the urine is being withdrawn through the catheter the stream is slow and lacks force, even when augmented by straining on the part of the patient. The urine may be clear, but in most cases pus and bacteria are present.

On cystoscopy no difficulty is encountered in passing the instrument. The bladder walls at the base, laterally and at the dome, show trabeculations. They are in most instances very fine, due to atrophy of the bladder musculature. At times they may be coarse, due to hypertrophy of those muscle fibers which have escaped being involved in the paralysis.

The trigone is not thickened as is the case in bladder obstruction, but is thinned and at both ends divides into trabeculae. The internal sphincter is relaxed, so that the cystoscope can be pulled into the posterior urethra without tilting it. The posterior urethra is ballooned, the walls are smooth and atonic and can be examined with an anterior scope. The mass of tissue composing the internal sphincter is seen rising like a wall in front of the supramontaine region, due to the relaxation of the muscles in the posterior urethra.

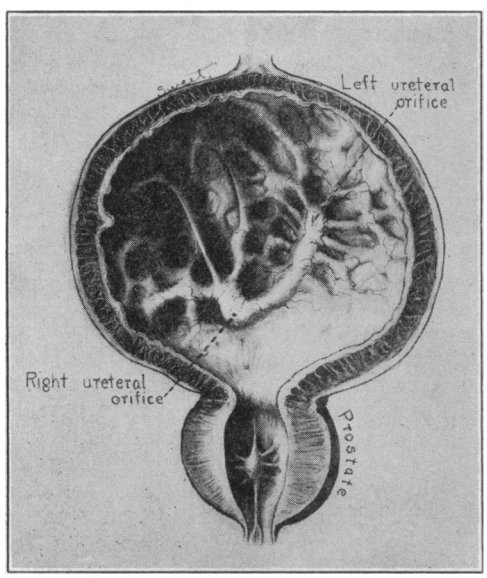
The cystogram shows a funneling in the region of the internal sphincter as a result of the filling of the posterior urethra from the bladder.

For the past eight months, we have had under our care a patient whose findings are similar to those just described and in whom none of the spinal cord conditions previously mentioned were found.

URINARY CONDITIONS AND POLIOMYELITIS

It is certain that the urinary changes in our case were due to poliomyelitis, and we believe that this disease should be considered as one of the possible causes of spinal-cord bladders.

The textbooks make little mention of bladder involvement in infantile paralysis. Strumpell[1] states that "micturition is sometimes a little disturbed at the beginning of the disease, but in most cases this disturbance completely disappears later." Osler[2] merely states that "the bladder may be involved." That there may be some effect on the urinary organs in the early acute stage has been noted by a number of clinicians. Schaller[3] cites a case of acute poliomyelitis in a patient twenty-one years of age in whom the bladder extended to the umbilicus and necessitated catheterization. In another instance the patient had to be catheterized twice because of acute retention. Peabody[4] in a summary of cases treated in the Boston epidemic of 1920 states that one patient was unable to void for two days. Bugbee[5] more recently reported the case of a boy one year old suffering with a distended bladder due to acute infantile paralysis. He states that this is the first case to be mentioned where bladder paralysis was the initial symptom.



Drawing showing the interior of the bladder with multiple cellules and distorted trigone. Note also the dilated posterior urethra.

Braasch[6] states that involvement of the centers of urinary control by poliomyelitis is not common. Occasionally, however, with extensive lesions, the motor fibers are affected, leaving an atonic bladder.

All the references which we have been able to find describe the bladder involvement as occurring in the acute stage of poliomyelitis. With one exception there has been no case history reported where the urinary tract was involved in the later stages of the disease.

Camphora[7] reported the case of a boy aged fourteen who was suffering from complete urinary incontinence for the last seven years. At the beginning of his illness gastro-intestinal symptoms kept the patient in bed nine months. Neither the boy nor his parents could remember whether or not there had been any convulsions or paralysis of muscles pointing to a participation of the spinal cord in the pathological process. The author concluded that as the patient had no symptoms of tabes, it may be presumed that the affection was poliomyelitis. This is the only report found suggestive of bladder involvement years after the onset of the disease.

REPORT OF CASE

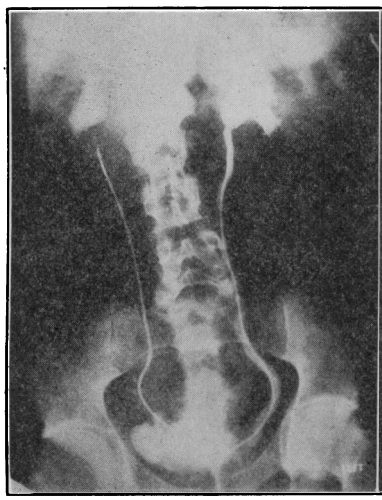
The case history of the patient to whom we have referred was obtained by questioning his mother, by reviewing the original notes of the late Dr. H. S. Sherman, kindly offered us by Doctor McChesney, and by examining the records at the Children's Hospital.

The history is as follows:

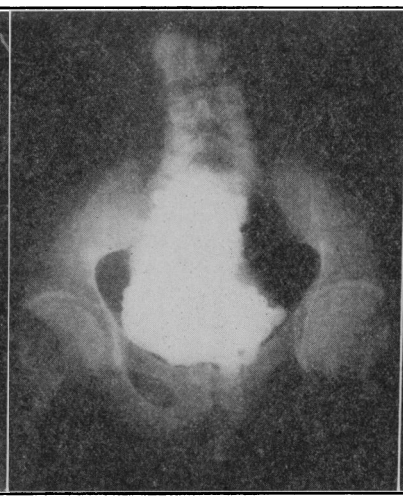
H. H., male, aged 31. Family history irrelevant. With the exception of the present illness, the patient has had only the usual diseases of childhood.

He was perfectly well until his second year, when he began to have pains upon stepping down suddenly. At this time he was irritable, did not sleep well, and had continual twitching in both legs and thighs, most marked on the right side. When 3 years old the twitchings became less marked, the pain disappeared and the general condition improved. His gait, however, was very poor and so at the age of 5 he was brought to Doctor Sherman, who stated at this time that the patient's condition was due to poliomyelitis.

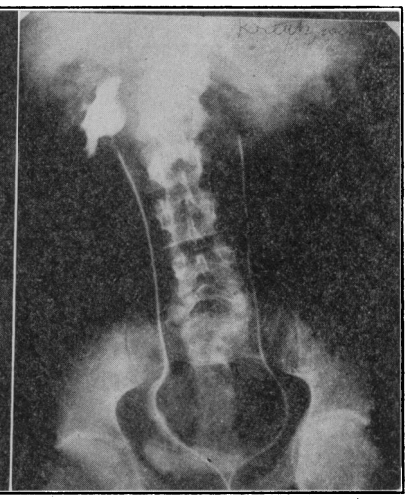
A tendon transplant was performed when the patient was 6 years old. The urinalysis at this time was



Left pyelogram—hydronephrosis



Cystogram showing multiple cellules and funneling of the posterior urethra.



Right pyelogram showing moderate hydronephrosis with obliteration of secondary calicies.

normal. The bedside notes give no indication of any bladder disturbance being present.

The next year the mother noticed the boy to be suffering with frequency, urgency, and bed wetting. He was brought to my father, who prescribed some powders.

A second tendon operation was performed at eight years. The hospital urinalysis at this time makes no mention of pus cells, but states that a great amount of detritus was present. The patient's urinary symptoms began when he was 7 years old, and have continued with little variation up to the present time. He now voids on an average of once every hour, has great urgency, hesitancy, and the stream is of poor force. Nocturnal enuresis occurs about two or three times a week. The urine has a foul odor. There is no dysuria or hematuria; no lumbar pains or ureteral colic. The sexual powers are greatly diminished, erections occurring only infrequently.

Physical examination shows partial paralysis of the muscles of both legs, associated with constant fibrillary twitching. The scrotum is pendulous and a left varicocele is present. The rectal sphincter is relaxed and there are some external hemorrhoids. The prostate is of normal size and consistency. The secretion shows nothing pathological. A voided urine specimen is alkaline; S. G., 1026; no sugar, heavy cloud of albumin. Microscopic examination shows no red blood cells, but a large number of pus cells. The bladder contains 265 cc. of residual urine. Cystoscopy shows the bladder floor, walls and dome to be covered with coarse trabeculations in the interstices of which are many cellules of varying depth. The trigone is greatly distorted and twisted to the left so that the right ureteral orifice is situated near the midline, while the left is far toward the left wall. The internal sphincter is greatly relaxed. No difficulty is experienced in moving the scope into the posterior urethra. The walls here are dilated, there are fine striations about the verumontaine, and the internal sphincter is seen rising up like a dam. The bladder urine showed B. coli. Number six ureteral catheters were inserted to each kidney pelvis without difficulty, notwithstanding the abnormal position of the ureters. Urine from both kidneys showed numerous red blood cells and an occasional pus cell. Cultures from both sides were negative. A half-hour phthalein test showed 14.3 per cent of the dye coming from the right kidney, while 21.8 per cent was eliminated on the left side.

Pyelograms showed moderate dilatation of both kidney pelves with beginning obliteration of the major calicies, especially on the right side. Cystogram showed a huge irregular bladder outline with a filling of the posterior urethra.

COMMENT

From the history it is quite certain that the urinary tract involvement began at the same time as did that of the spinal cord. It is also evident that the same agent which produced a paralysis of the nerves going to the leg muscles had also caused a paralysis of the sympathetic and parasympathetic nerves which supply the bladder.

Whether or not the hydronephrosis is part of the same process, we do not know. It is not due to back pressure from the residual urine, as the kidney cultures were negative and the cystogram showed no opaque solution in the ureters or in the renal pelves. This is in keeping with Plaggemeyer,[8] who found that in cases of gunshot wounds of the spinal cord there was no back pressure from the residual urine.

The dilatation may be due either to paralysis of the nerves supplying the kidneys or to the abnormal position of the ureters as they enter the bladder. We believe the latter to be the most likely cause.

Plaggemeyer[8] in all his cases of bladder paralysis due to spinal-cord injury found no vesical stones notwithstanding the constant residuals, the majority of which were infected. It is interesting to note that in the case reported here no vesical calculus was observed although the patient has had infected residual urine for at least twenty-four years.

The symptom of nocturnal enuresis should be considered of great significance by urologists. It is possible that in patients in whom this occurs there may be some early changes in the nervous system which can be recognized by the cystoscope. If, in these cases of bed wetting, the bladder walls show fine trabeculations and perhaps beginning relaxation of the internal sphincter a thorough neurological examination should be made.

The treatment of spinal cord bladders depends entirely upon the pathology found in the central nervous system. The bladder treatment of patients suffering with traumatic injury to the cord

is entirely different from that where the cord condition is due to some disease.

As a result of the sudden complete bladder paralysis produced by an injured cord, infection occurs easily and for that reason catheterization should not be performed. There may be acute distention for a few days, causing great distress to the patient, but in time the automatic bladder will develop: the patient will be quite comfortable and no infection of the urinary tract will result.

Since it is the ascending urinary infection which ultimately proves fatal, no instrumentation whatsoever should be performed upon a non-infected bladder.

On the other hand, if the disease in the cord is amenable to treatment, we should regard the bladder condition as a secondary one which will improve with the proper treatment after the primary lesion has been removed. In such cases the aim should be to keep the bladder clean and to retain its tone. If there is infection present the patient should be catheterized several times a week, the bladder should be washed with some antiseptic solution, and then some silver salt should be instilled. Urotropin given at intervals is of benefit in cases having large amounts of residual urine.

Muscle tone can be increased by instructing the patient to empty his bladder as completely as possible and to start and stop the stream at frequent intervals while voiding in order to strengthen those muscles which are not paralyzed.

This treatment has been of benefit in our patient. The nocturia and bed wetting have stopped entirely and the bladder urine shows very little pus. The residual is not much less, but urination is performed at the same hour every day, and in this manner no discomfort is experienced.

CONCLUSIONS

From a study of the case presented here and a review of the literature we have reached these conclusions:

1. In the early stages of poliomyelitis acute retention is sometimes a complicating factor.

2. Gross changes in the urinary tract may occur in chronic poliomyelitis which will give rise to the typical findings characteristic of spinal-cord bladders.

1195 Bush Street.

REFERENCES

1. Strumpell: Textbook of Med., Vol. ii, 1914, p. 405.

2. Osler: Textbook of Med., 1912, p. 366.

3. Schaller: Calif. State Jour. of Med., Vol. xx, No. 4, April, 1924.

4. Peabody: Boston M. & S. J., Vol. 185, No. 6, August 11, 1921, pp. 174-76.

5. Bugbee: Jour. of Urology, Vol. xiii, No. 3, March, 1925, pp. 333-35.

6. Braasch: J. Urology, April, 1925, Vol. xiii, No. 4, pp. 383-98.

7. Camphora: Gazette degli Os pedali, Milan, 33, 1593, December 19, 1912.

8. Plaggemeyer: J. A. M. A., Vol. 73, No. 21, November 22, 1919, pp. 1599-1606.

OCULAR MELANOSIS—ITS SURGICAL AND RADIUM ASPECTS*

CASE REPORT

By P. OBARRIO, M. D.
AND
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MRS. H———, a seamstress by occupation, 56 years old when she first consulted us, February 1, 1923.

History—She tells us that since about the age of 24 she had noticed a black spot on the white of her right eye. This spot had the peculiarity that it would occasionally disappear only to reappear again possibly a little larger in size. These changes would take place without any treatment whatever.

Her family history, personal history, blood examinations, urine and all the other antecedents were negative. She was a well nourished, healthy person.

Condition When First Seen—On the superoexterior quadrant of the right eye and encroaching on the corneal margin there was a tumefaction of the conjunctiva of about 6x4x3 mm. with possible attachments to Tenons capsule and the sclera.

The growth was highly pigmented and somewhat movable.

All of the conjunctiva, both bulbar and palpebral, was so highly pigmented that this pigment could be easily rubbed off with a bit of gauze or cotton.

The cornea, refractive media and the fundus were normal.

Refraction as follows:

R + 50 C. ax. 135° 20/20
L + 75 S. + 25 C. ax. 130° 20/20

TREATMENT

Under local anesthesia this growth was removed, the conjunctiva undermined to procure sliding flaps, and sutures were applied.

Shortly after this a very considerable portion of the pigment of the conjunctiva had disappeared.

Recurrence—It was not long before a secondary growth made its appearance, this time at the lower cul-de-sac.

During the development of this second growth the universal increase of pigment about the conjunctiva seemed to be in direct ratio to the extent and duration of the growth.

This growth was likewise extirpated only to recur once more in the immediate vicinity.

We felt at the time that as each extirpation entailed the removal of a considerable portion of the conjunctiva, that a repetition of this procedure would produce a vexing entropion through cicatrical contraction.

The patient did not return to us for a considerable period of time after the second removal of the growth. When she eventually returned, February, 1926, the condition of the eye was worse than ever; not only was there a recurrence of the growth at the lower cul-de-sac, but the pigmentation of the

* Read before the Eye, Ear, Nose, and Throat Section, California Medical Association, at the Fifty-Sixth Annual Session, April 25-28, 1927.